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ART. I.—CASE OF REPEATED ATTACKS OF APOPLEXY WITH APHASIA.

BY DR. H. D. SCHMIDT,
PATHOLOGIST OF THE CHARITY HOSPITAL OF NEW ORLEANS, AND MEMBER OF THE AMERICAN NEUROLOGICAL ASSOCIATION.

(*WITH PLATE.*)

FOR a number of years, *aphasia*, that particular affection of the brain, which, in many cases, is found associated with, or directly resulting from cerebral apoplexy, has attracted the special interest of pathologists. The following case, which occurred in my private practice, and which terminated fatally about a year after the first cerebral disturbance, offered me ample opportunity, not only to closely observe and study the phenomena of the disease during life, but, moreover, to compare them with the results of an accurate and minute examination of the brain after death. It is for this reason that I consider it worthy of record.

On the morning of November 11th, 1876, I was called to Mr. F——, a gentleman of about fifty years of age, and with

whom I had been acquainted for more than thirteen years. I found him in a condition presenting symptoms of severe cerebral congestion. As I learned from a later account of the patient himself, he was taken by the disease during sleep, after having retired to bed at midnight, worn out by the duties of his business on the previous day; and without the knowledge of his family, who found him in this condition, when entering his room in the morning, not long before I was called. When I first saw him, he was in a state of partial consciousness; his tongue, swollen and almost black from congestion, was protruding from the mouth and tightly held by the teeth; and, in consequence, the patient was unable to articulate any word. There was left hemiplegia, with slight paralysis of the muscles of the inferior part of the face, the tongue, and the levator palpebræ on the same side. In examining the heart, a very slight murmur could be detected. From the position in which the patient had been found, and, also, from the appearance of the bed, I judged that the attack had been accompanied by convulsions. After a few days, the complete consciousness of the patient returned. The paralysis, also, gradually subsided, and about the ninth day from the commencement of the attack, he was able to attend to his business. With the exception of the skin covering the internal half of the left arm, and which presented a bluish color, similar to that of a bruise, no further trace was left of the disease. As this portion of the skin had experienced no violence, the bluish color must have been owing to a neuro-paralysis of the minute vessels of the skin, a condition which soon after subsided. Some facts which I observed during this attack, to be spoken of hereafter, led me to suspect disease of the arteries of the brain as the cause of this congestion; and, therefore, I cautioned the patient particularly against any excess in eating and drinking, or any other excitement. Nevertheless, a second attack, invited, perhaps, by an imprudent act of the patient, occurred on February 16th, 1877. His sense of hearing had been slightly affected for a number of years, but, possessing his full share of vanity, he tried to hide this defect as much as possible from the world. On the day previous to this second attack, he took a sudden notion to consult an aurist,

with whom he was acquainted, and who, not knowing his previous condition, passed an electrical current through his head. After this operation he passed a sleepless night, and, on the next day, felt so badly as to leave his office early in the afternoon and start for home. While in the street-car, he was seized with vertigo, but kept sufficient strength and consciousness to leave the car, and also reach his house, where he was prevented from falling to the ground by his wife, who had opened the door. This time he was struck with hemiplegia of the limbs on the right, and paralysis of the muscles of the inferior part of the face, the tongue, and levator palpebræ on the left side. The paralysis of the face must have occurred before he reached the house, for his face was distorted when his wife first beheld him. With difficulty he was put to bed, and I was called. I found him in the condition already stated, laboring under a partial stupor, though sufficiently conscious to understand what was said to him. However, he could not speak; he was affected with true aphasia; for although his tongue was drawn to the right side, he was able to protrude it and draw it back. The rapidity of the pulse was slightly increased, about ninety per minute, but it was rather weak. The respiration was peculiar; being normal for some minutes, it gradually became weaker, and, apparently, ceased altogether for from five to ten seconds. Then the thorax recommenced to expand, and, while the patient was heaving for breath, the respiration, accompanied by a slight stertor, became forced, after which it slowly returned to the normal standard. When asked, the patient complained of pain in the head, particularly in the parietal and frontal region of the right side, directly above the temples. When not disturbed he remained in a drowsy condition, from which he was, however, roused, from time to time, by the irregular performance of the respiratory function. At no time during this second attack, was there a complete loss of consciousness,—a phenomenon which I supposed to have existed during the first stage of the first attack, coming on, as will be remembered, during the sleep of the patient.

As regards the remedies immediately applied during this second attack, I will only state that they were the same as had

proved successful during the first, namely: cold to the head, leeches to the temples, and, afterward, the administration of bromide of sodium, with the addition of good nourishment in the form of essence of beef, milk, etc. However, if I had known the true condition of the minute, as well as that of the larger arteries, which the post-mortem examination afterwards revealed, but which, at this time, I only faintly suspected, I would have omitted the leeches, and probably, also, the bromide of sodium, and have contented myself with the application of cold to the head. But, as the first attack had passed over so favorably, and finding in this second one no loss of consciousness, I regarded the symptoms as those of hyperæmia, accompanied, perhaps, by a serous effusion into the subarachnoid space and the ventricles, which, by compressing the brain, might be the cause of the pain and feeling of fullness in the head, and, also, of the respiratory troubles.

On the next morning I found the patient slightly better. Although the respiration had greatly improved, being now regular, he was, nevertheless, lying in a state of drowsiness, from which he was aroused when he was addressed. He also made some attempts to answer; but, as he used wrong words of expression, he could not be understood; and the only way of ascertaining his wishes was in putting questions to him in various ways. At this time his language consisted only of one word, which was "paper."

Thus the case went on without much change until the fourth day, when, again, the respiration became more irregular, and the drowsiness also increased, even so much as to induce me to doubt a favorable issue. Not having observed any particular benefit derived from the bromide of sodium, I discontinued its use, and relied solely on rest, careful nursing and cold to the head, with special attention to the secretory and excretory functions; and to my satisfaction, the case took a more favorable turn; for the patient slowly improved in strength. But as the drowsiness disappeared and the mind became clearer, the symptoms characterizing aphasia were rendered more prominent. About three weeks from the first day of this attack, when the patient was able to sit up for a few hours, it was discovered that he had not only lost the faculty of express-

ing his ideas by articulated language, but he had also lost the knowledge of reading and writing. From this time on, however, an improvement gradually manifested itself in the paralyzed limbs; first in the lower, and then in the upper one; and, somewhat later, also in the face. The same was the case with the faculty of speech; though here the improvement consisted only in the recollection of the most common-place phrases, such as: "Good evening," "how goes it," "first rate," etc., for, as soon as the subject of the conversation was changed, the patient became confused and unable to continue it, unless the words which he endeavored to find, were, so to say, put upon his tongue. With the exception of the pain in the parietal and frontal region of the right side, which still persisted, the general health was as good as it could be expected to be. The circumstance of the pain in the head being located on the right side, induced me to regard this case as deviating from the general rule, though the patient was right-handed. About the middle of April, the paralysis of the limbs and face, with the exception of the levator palpebræ, had sufficiently disappeared to enable the patient to dress himself, and walk about the room; and soon afterwards he could leave the house and take a ride. At this time, also, he could write his name again, and read the heading of newspaper articles, printed in large letters. In fine, excepting a slight pain in the head, which still persisted, he felt well; his mind also seemed to be more invigorated. But, though he spoke more fluently and with less interruption; though he could read and understand insignificant articles in the newspapers, he still became confused and stopped short in the conversation as soon as it turned upon a subject whose discussion required more than ordinary phrases.

Upon my advice, based upon the principle of perfect rest of the mind, in order to give nature a chance of repairing the damages done, all visitors, though his personal friends were numerous, had been cautiously kept away from the patient during his illness; and having thus far witnessed the restorative powers of nature, I advised him not to re-enter society for some time, but to take his abode in the country as soon as the season would permit. In the beginning of May, therefore,

he went to a secluded place, free from all causes of excitement, where he lived in the house of a small farmer until about the middle of June, when he returned to New Orleans, and paid me a visit. He had gained in flesh, and his general health was excellent. His speech, also, had improved, so much, even, that a careless observer not acquainted with the circumstances would not have detected the defect at once. But, notwithstanding these improvements, whenever the conversation was continued for any length of time, the aphasia still appeared. A few days afterwards he went to "Point Clear," a quiet watering place with a good hotel on the shores of the Bay of Mobile, where, besides the excellent breeze coming from the gulf, he could also enjoy the stimulating effects of sea-bathing. There his condition improved so much, that, in the beginning of September, longing for more society, he returned to New Orleans.

The time which I had always feared, had now arrived: the time which certainly would test the stability of his recovery. His general health was still excellent; he was able to read, and also to write; but, as I had predicted to his family, not sufficiently well for the discharge of his daily duties. He could speak, but not enough to re-enter the active life of a cotton broker, a branch of business in which he had been engaged for many years. He felt his incapacity, and resigned his business. By the exertion of his many friends he received the office of supervisor in a cotton press; and all seemed to go well until one morning, about the middle of October, when, while dressing himself, he suddenly lost his consciousness, and fell upon the floor. When I saw him, soon after the attack, he had recovered his mind and felt well; and, on the next morning, went to his business as usual. On the morning of November 12th, at the moment of having finished dressing himself, and ready to leave the room, he fell again to the ground with loss of consciousness. I found him in bed; he had already recovered his consciousness. Excepting a few bruises, there were no alarming symptoms present; and, after two or three days, he resumed his daily occupation, until, finally, on November 21st, he was seized with the fatal attack, accompanied by all the symptoms of extensive cerebral hem-

orrhage. He had left his home in the morning, apparently well; but, about 2 o'clock P. M., while engaged at the press, he commenced to feel ill, and refusing to send for a carriage to take him home, he placed himself upon a chair in such a position as would afford him rest, and at the same time secure him from falling to the ground. Very probably he was seized with drowsiness soon after, for at 4 o'clock he fell from the chair in a state of unconsciousness. At 6 o'clock, his son, who had been called to the scene, brought him home. When I saw him, soon after, he lay in a state of deep coma, with a slow and full pulse, and stertorous respiration. The lid of his left eye was considerably swollen and discolored from a severe bruise of the skin of the superciliary region, upon which he had fallen. The pupils were contracted. There was no anæsthesia of the skin; on the contrary, irritation of the skin was followed by reflex action of the muscles. He moved his hand, and I was unable to detect any paralysis of the limbs. On the next morning, however, paralysis of the limbs on the left side could be easily detected; for, while the right hand was in motion from time to time, the left arm lay lifeless and never moved again. With the exception of the respiration becoming gradually weaker, more labored, and more stertorous, he remained in this unconscious condition until November 24th, at three o'clock a. m., when he died. For the interest which I had taken in the case I obtained the permission of the family to examine the brain. The results of this examination were as follows.

Post-Mortem Examination of the Brain.—When the brain was removed from the cranial cavity, an effusion of blood, partly fluid, partly coagulated, and amounting to about four fluid ounces, was found at the bottom of the cavity. There was nothing abnormal in the appearance of the dura mater. The examination of the pia mater showed at once the original cause of the disease,—degeneration of the blood-vessels. While most of the veins were filled with blood, a great number of the arteries, especially the larger ones, and not only those of the cerebrum but also some of the cerebellum, were affected with atheromatous disease. This degeneration was so general that it was even found upon the smaller branches, as

far as they could be distinguished by the naked eye. On the basilar artery, the intervals left between the atheromatous swellings or knots hardly measured more than one-fourth of an inch. The invasion of the disease upon the anterior, middle, and posterior cerebral arteries equalled that on the basilar, for, though the intervals in some places were of greater length, in others the dilatations of the vessels approached each other closely. A considerable number of the smaller arteries of the pia mater were filled with blood. In searching for the ruptured blood-vessels, from which the blood found in the cranial cavity might have escaped, two large, dark-brown or almost black-looking spots, presenting an ulcerated or lacerated appearance through the pia mater, were found upon the surface of the temporal lobe (Fig. 1). The one was situated in the middle temporal convolution, about two inches behind the apex of the lobe, and measured 7 x 9 mm. in diameter; the other, situated in the inferior temporal convolution, measured about 6 mm. in diameter.

After this macroscopic examination, the pons Varolii, medulla oblongata, and cerebellum were separated from the cerebrum by a horizontal section through the crura cerebri, directly above the pons; and then a transverse vertical section was made through the cerebrum, directly behind the tuber olfactorium. This section, passing through the corpus striatum, nucleus lentiformis, and claustrum of each hemisphere, revealed a large apoplectic cyst (Fig. 1), extending throughout the whole island of Reil of the left hemisphere. When the section was made, a moderate quantity of a brownish, rusty-colored fluid escaped from the cavity. After the escape of this fluid, the latter appeared collapsed, its walls almost touching each other. The cavity itself was lined by a distinct pseudo-membrane, about ½ mm. in thickness. As this membrane was of a considerable consistence, and slightly elastic, it permitted itself to be separated from the adjacent structures without being torn. The inner surface of the cavity represented by the membrane, was rendered uneven by the presence of little elevations and depressions, and had a muddled appearance; in some places of a reddish, and in others of a lighter or darker brown color. The cyst, which was most probably

one of long standing, as will be seen directly, presented in the section the form of a slit, 3 ctm. long, and occupied, as the drawing shows, the place of a considerable portion of the claustrum and the capsule; the nervous substance, adjacent to its inferior third, was in a condition of softening, extending on its median side into the nucleus lentiformis, and laterally into the white substance of the island of Reil. A smaller cyst, about the size of a small pea, was found in the white substance of the same section, directly below the superior frontal sulcus, at the place where the white substance of the superior frontal convolution meets with that of the middle frontal convolution (Fig. 1). This cyst was not lined by a pseudo-membrane, but was probably formed by a process of softening of this substance. A portion of the latter, between the larger and smaller cyst, was also in a state of softening.

A transverse vertical section of the same hemisphere, ½ ctm. anterior to the island of Reil, revealed the existence of a third apoplectic cyst, also lined by a pseudo-membrane of the same nature as the one above described. This cyst represented a narrow cavity of an almost rectangular form (Fig. 3). Commencing in the apex of the superior frontal convolution, it extended along the margin of the gray substance, forming the median wall of the superior frontal sulcus, and, in entering that substance at the bottom of the sulcus, bent in nearly a right angle, and passed through the white substance of the middle frontal convolution. That part of the cyst contained in the superior frontal convolution, exceeded in length and depth the remaining portion in the adjoining convolution. Its length, from its beginning at the apex of the convolution to the bottom of the sulcus measured 23 mm.; the entire depth of this part of the cyst, in an antero-posterior direction, was 2½ ctm. The remaining part of the cyst in the adjoining convolution was quite shallow.

A third transverse vertical section, passing through the splenium of the corpus callosum, and through the lateral ventricles directly behind the commencement of the descending cornua, disclosed two more cysts in the same hemisphere; they were not lined by a pseudo-membrane, but formed by softening. The larger one, of an irregular rhomboidal form,

measured 11 mm. in length, and 8 mm. in width, and was situated between the bottom of the interparietal sulcus and the lateral wall of the left ventricle, extending beyond the apex of the latter. The smaller one, about 6 mm. in length, and 3 mm. in width, was situated in the median wall of this ventricle.

After this preliminary examination, the different portions into which the brain had been divided by the sections of the knife were put, for preservation, into a solution of bichromate of potassa—of a strength about two ounces of the salt to ten pints of water—to be subsequently examined with more leisure.

On the following day the pia mater was carefully removed from all the pieces resulting from the sections of the brain above described. But, in order to keep the cut surfaces of the different pieces into which the brain had been divided as level as possible, I determined to make no farther sections until they had, by the action of the preserving fluid, obtained sufficient consistency to preserve their form. Meanwhile I proceeded with the microscopical examinations.

In the right hemisphere, as may have been noticed, no lesion whatever had thus far been discovered by the sections made. But when, about a week afterwards, the middle and larger portion of that hemisphere was again divided by a transverse vertical section, passing about 4 mm. in front of the opening of the aqueduct of Sylvius into the third ventricle (Fig. 2), an enormous apoplectic cyst, the largest of all, was discovered. At the level of the section this cyst measured 3 ctm. in length, and 1½ ctm. in width, reaching in its vertical direction, as the drawing will show, from the nucleus tegmenti into the highest portion of the nucleus lentiformis, and to a level with the posterior, or caudated portion of the corpus striatum. Posteriorly, the cavity extended throughout the whole posterior portion of the thalamus opticus, the pulvinarium, leaving nothing of this ganglion but its cortex, in the form of a thin shell; anteriorly it extended, in a somewhat lateral direction, about as far as the location of the anterior commissure; thus having destroyed the greater part of the thalamus opticus, a portion—perhaps the larger also—of the basis and tegmentum,

together with a portion of the nucleus lentiformis and the claustrum. The antero-posterior diameter of the cavity was about 3½ ctm. This extensive cavity was almost entirely filled with coagulated blood, sufficiently consistent to be cleanly cut by the knife in making the section; only a small portion of the blood escaped from the cavity in a fluid condition. After all the blood was removed by a gentle stream of water, it was found that the cavity must have been of recent date, as its walls were not lined by a pseudo-membrane, but quite soft. In looking for the original seat of this hemorrhage, a group of numerous punctiform apoplectic foyers were discovered in the substance of the tegmentum, along the inferior border of the cavity. It is obvious that the hemorrhage had arisen from these minute centres, and some time may have elapsed before the amount of effused blood was large enough to destroy the neighboring tissues by its pressure and form a cavity of such an extent. But, very probably, other minute hemorrhages occurred at the same time in the tissues already destroyed, the remains of which, in a softened condition, had been washed away with the clot of blood. As regards the latter, it is difficult to determine whether the coagulation had taken place before the death of the patient, or whether it may have been caused by the action of the preserving fluid. The white substance between the upper half of the claustrum, the nucleus lentiformis, and the cauda of the corpus striatum, near the upper border of the cavity, was also undergoing the process of softening (Fig. 2).

The condition of the larger arteries and of their subordinate branches, as far as can be ascertained by a macroscopic examination, has already been stated; and, as the pathological process of their atheromatous degeneration is so well understood, it requires no further remarks. But, as regards the more minute vessels of the pia mater, a microscopical examination of small pieces of this membrane showed that the veins were filled with blood, while the arteries were empty. The greater number of the latter appeared normal, showing their component anatomical elements distinctly. A smaller number, however, appeared pale and faint, and also much contracted, with a caliber scarcely as wide as the thickness of their coats, and

almost too small for the passage of one row of blood corpuscles; they were entirely empty. This pale appearance may, perhaps, have been owing to a commencing atrophy, caused by the partial or total occlusion of one of the larger vessels.

In examining small portions of the softened nervous substance, as well as others taken from the substance in the neighborhood of the apoplectic cysts, or of the punctiform hemorrhages, microscopically, it was found that the greater number of minute blood-vessels, the capillaries, and particularly those vessels intermediate between the latter and the arterioles, and appearing to consist of only two coats, were undergoing degeneration. The degenerative process here commenced with the nuclei in the walls of the vessels. Those small traces of granular protoplasm, namely, which in the normal condition of the vessel may be detected upon the surface of these nuclei, abnormally increase in quantity; and it is this accumulation of protoplasm, causing an uneven thickness of the wall of the vessel (Figs. 4 and 5), which constitutes the first stage of the degenerative process. If the degeneration goes on further, the accumulations of protoplasm gradually undergo a fatty metamorphosis, giving rise to the final rupture in the walls of the vessel. These accumulations of protoplasm of course encroach upon the caliber of the vessels in proportion to their extent; and, if a large number of vessels are affected, they must finally seriously interfere with the capillary circulation of that region. In this manner minute thromboses will be formed in the capillary vessels; especially when nuclei, placed very near to each other, become affected, an occurrence which may result in the occlusion of the vessel. In the case under discussion I directly observed some of these capillary thromboses where the vessels in question were filled with a row of blood corpuscles, reaching to each side of the thrombosis. In the smaller arterioles, also, the degeneration may finally result in the occlusion of the vessel, as seen in Fig. 5. But, even if this does not occur, the diminution of the caliber and the unequal width of the vessels, together with the uneven condition of their inner surface, caused by the projection of the accumulated protoplasm, must eventually be followed

by a considerable disturbance of the circulation. Although, in this case, the fatty degeneration of the protoplasm had as yet not progressed as far as in other cases which I have observed, it was, nevertheless, sufficient to give rise to a softening of the mass, manifesting itself in the form of minute aneurisms (Fig. 4), and to terminate in a rupture of the walls, followed by an extravasation of blood.

It appears that the effusions of the blood found in the right hemisphere, and producing the cavity in the thalamus opticus, etc., as well as the other found at the base of the cavity of the cranium and coming from the temporal lobe, had their origin in capillary hemorrhage. A microscopical examination of the minute apoplectic foyers in the tegmentum (Fig. 2), showed that they were of quite a recent date, for though their margins were so well defined as to allow an easy separation from the surrounding tissues, they were, nevertheless, destitute of those fibrinous capsules by which they are, in many cases, ultimately surrounded if the patient survives the accident,—and time is given to the organization of the fibrin contained in the drop of blood which escaped from the vessel. The centres of these minute foyers appear to the naked eye somewhat lighter than the rest, a phenomenon which is probably owing to the colorless corpuscles accumulating there. The fine fibrillæ of the neuroglia, projecting beyond the periphery of those foyers separated from the surrounding tissues, appeared very distinct. In some instances the blood escaping from the opening in the vessel does not accumulate to form a single drop, but encases the little vessel by traveling for a certain distance along the surface of its wall; and, in others again, the blood is imbibed by the neighboring tissues. Such conditions, however, can only be revealed by microscopical examination.

As regards those two dark looking spots of an ulcerated or lacerated appearance, found in the left temporal lobe, and from which the hemorrhage into the cranial cavity had arisen, it was, after the careful removal of the pia mater, found that while the one represented the opening of a cavity the other was only a superficial lesion. The first (Fig. 1), situated in the gyrus temporalis medius, led into a cavity of about 4½ ctm. in length, and 2 ctm. in width, extending throughout the white

substance of this convolution. The entrance was filled with a dark brown mass, resembling an old clot of blood, but which a microscopical examination showed to consist of minute blood-vessels in the state of degeneration, as above described, and, also, of broken down nervous tissue. The whole mass extended into the cavity,—the course of which will be found indicated in the drawing by dots—filling up its middle third. The second lesion, a little smaller in diameter, was situated in the gyrus temporalis inferior, about one inch posteriorly to the former; it was entirely confined to the cortical layer, and filled like the first, with broken down nervous substance and diseased blood-vessels.

The microscopical examination regarding the structure of the pseudo-membrane, was interesting, as it distinctly showed that this membrane was entirely formed from the component elements of the original clot of blood. It consisted of an amorphous substance or matrix, in which a number of other elements were found embedded. Small portions of this substance, torn with fine needles into minute fragments, appeared to consist of minute irregularly oval or round bodies of a greenish tint, fused into minute irregularly shaped masses of various sizes, and held together by a hyaline substance (Fig. 6, *a*). Through this substance a moderate number of delicate and smooth fibrillæ were seen to pass. From a careful examination of these elements I am inclined to regard them as the colorless remains of the colored blood corpuscles of the clot, fused together by mutual adhesion into irregularly shaped groups or masses of different sizes, and the whole united by the hyaline substance—very probably derived from the liquor sanguinis of the clot—into the form of a pseudo-membrane. And this view seemed to be corroborated by the presence of numerous other bodies, mostly round in form, but differing much in diameter and, also, in color. These bodies (Fig. 6, *b*), were spherical aggregations of larger or smaller granules, being evidently the remains of the colored blood corpuscles, deprived of their coloring matter, and represented by their protoplasm only. Besides these bodies, a considerable number of individual isolated corpuscles were also observed, some of which, larger than the others, still showed a

double contour. A number of fine granules and molecules were also observed. The hæmatin, escaped from the colored blood corpuscles, was distributed, either formless or crystalized, throughout the membranous mass. Perhaps the greater number of these aggregations, formed by the protoplasmatic remains of the colored blood corpuscles, were colorless, possessing only a pale greenish tint; but there were others, especially the smaller ones, which, colored by the free hæmatin, appeared in various shades, from the light yellow to the dark brown or black. Some of the darkest seemed to be covered by a layer of very minute hæmatin crystals. The hæmatin, besides coloring these bodies, was also found both in the form of larger or smaller patches and in that of crystals of different sizes (Fig. 6, *c*); some of the latter appearing as minute dark molecules.

Besides the elements just described, there were also a number of minute blood-vessels met with in the pseudo-membrane. Some of them appeared almost unchanged in structure, and filled with blood corpuscles, while the greater number appeared very pale, being evidently in a state of disintegration; even blood corpuscles were observed in some of the latter, though with very pale outlines. The blood-vessels found in the membrane were most probably identical with those which formerly belonged to the nervous substance, displaced or destroyed by the presence of the clot. But while the greater number were obviously undergoing pathological changes, it appeared that others, filled with blood, had preserved the integrity of their structure.

A microscopical examination of the outer surface of the membrane attached to the adjacent nervous tissues, showed the latter in a condition of disintegration, a circumstance explaining the easy separation of the membrane, as stated before.

The microscopical examination of the walls of the large cavity in the right hemisphere, which, after being washed, still preserved an appearance, muddled, more or less, by brownish spots, owing to the diffusion of blood corpuscles throughout the nervous tissue, showed this tissue in a state of disintegration. It is the medulla of the nerve fibres which

seems to be most prone to decay, while the axis cylinders, and also the ganglionic bodies, consisting of protoplasm, offer the greatest resistance. Accordingly, the elements met with were fragments of very fine nerve fibres, either in a varicose form, or, deprived of their medulla, represented only by the naked axis cylinders. A number of larger axis cylinders, destitute of the medulla, were also observed. Some of the ganglionic bodies presented a natural appearance, while others appeared more pale, but the processes of most of them were torn. All these elements, together with fragments of diseased, or also, normal minute blood-vessels, usually filled with blood, were mingled with the disintegrated granular substance of the ganglionic nervous matter. A considerable number of more or less round patches with defined contours were also observed; they were fragments of granular substance, showing distinctly the minute groups of granules of which it consists.

Remarks.—The comparison of the clinical history with the results of an accurate post-mortem examination of the preceding case, reveals some points of interest relating to the pathology and, perhaps also, the treatment of organic cerebral disease worthy of a careful review and study. The cerebral hemorrhages, occurring at different times in this case, were evidently due to the existing disease of the smaller blood-vessels, and, from some similar cases which I have examined as thoroughly as this one, I am inclined to think that in the great majority of cases of cerebral hemorrhage, the hemorrhage, with disregard of its extent, is of a capillary origin. At any rate it may be asserted, that in all cases of cerebral apoplexy, not terminating fatally at the first attack, the hemorrhage is due to a rupture of capillaries, or of those vessels intermediate between the latter and the arterioles. A hemorrhage caused by a rupture of one of the larger cerebral arteries, or of one of their subordinate branches, must necessarily be fatal, as the force of the current of the blood through these vessels certainly predominates over the inherent power of coagulation possessed by the fibrin of the blood. Neither can the vessel close by its own contraction if the rupture was the result of a degeneration of its walls. The process of fatty degeneration of the arterial walls, commencing in chronic

endo-arteritis, is generally slow in its course—especially if accompanied by calcareous deposits—and a rupture of the degenerated artery would hardly take place without—in consequence of its degeneration—a preceding disturbance in the circulation of that part of the organ which it supplies with blood. But, as any irregularity in the performance of the circulatory function must necessarily affect the nutrition, not only of the nervous tissues, but also of the capillaries and arterioles, and induce or hasten in these minute vessels the degenerative process,—it is more probable that a capillary hemorrhage takes place before the degenerated portion of the larger vessel may yield to the pressure of the current of the blood produced by every contraction of the heart. However, as certain diseases affect only certain tissues, or tissues related to them, and as the endothelium of the inner coat, the *intima* of the larger blood-vessels, is almost identical with the delicate transparent walls of the capillaries, it is quite possible that the same process of degeneration may simultaneously take place in both kinds of vessels. Or, it may also occur that the minute blood-vessels be seriously affected, while the larger arteries preserve their integrity.

In the clinical history of this case I stated that I regarded and treated the symptoms accompanying the first attack as those of simple hyperæmia of the brain; and the rapid amelioration and disappearance of these symptoms seemed to speak in favor of this diagnosis. But since the true condition of the brain was revealed by the post-mortem examination, the correctness of that diagnosis may reasonably be questioned, as to whether these symptoms might not be attributed to a capillary apoplexy of a limited extent.

From the examination of some other cases I have reason to believe that capillary hemorrhages of small extent may take place in the brain, without being followed by symptoms of a serious character, such as a paralysis persisting for any length of time, or even complete loss of consciousness; the second attack of the present case also proves this assertion. It is possible, therefore, that the first attack depended on a similar cause. Moreover, as during this attack the paralysis of the limbs—regarding that of the face, being on the same side,

as an exception to the rule—was confined to the left side of the body, it is possible that the seat of the hemorrhage was in the right cerebral hemisphere, and in the same parts,—the thalamus opticus, tegmentum, basis, &c., which, during the last and fatal attack, were destroyed by a more severe hemorrhagic extravasation. The probability of this occurrence becomes more apparent by the fact of the paralysis of the limbs being on the same side in both attacks; and, from the great destruction of tissue, caused by the last hemorrhage in these parts, and the large cavity left behind, we may presume that, at a previous time, a capillary hemorrhage of limited extent may have occurred in the same place, preluding the last or fatal one.

In the beginning of this article I alluded to a certain fact observed, which had induced me to suspect a diseased condition of the blood-vessels of the brain already during the first attack. In examining the patient, namely, I noticed a considerable enlargement and very tortuous course of the anterior branch of the left temporal artery, sufficiently marked to render the vessel very prominent and to project considerably above the level of the skin. Until that time I had never directed any particular attention to this phenomenon, though it is frequently met with, and is owing to a dilatation of the vessel, indicating disease of its coats, and resembling in its nature an aneurism. From this fact observed I then presumed that some of the vessels within the cranium might be similarly affected, and, in some way or other, been involved in the existing hyperæmia. These suspicions were strengthened by meeting some weeks later a similar case, presenting the same phenomenon. This was a gentleman connected with one of the daily newspapers here, about forty years old, and whom I had known for nearly ten years. Some time previous to his visit to my office he had been struck with hemiplegia of the right limbs and paralysis of the face on the left side, which his physician had declared to be of syphilitic origin. The correctness of this diagnosis, however, I regarded as doubtful, as the patient assured me that, besides a small sore on the penis in his youth, he had never noticed any symptoms of syphilitic disease upon his body afterward. In this case, also,

I observed the dilated and tortuous condition of the anterior branch of the temporal artery, but on the right side of the head. In directing his attention to this fact, I learned that he had never taken notice of it before. With the exception of his right hand and of the muscles of the face, the paralysis had disappeared; and, though he was again able to write, hand and brain were soon fatigued by the exertion of the performance. Once more he paid me a visit, and then I saw him no more. But, about two months afterward, I learned from the newspapers that he had died from another attack of apoplexy, and that he had been found by the neighbors, who were called to the scene, on the floor, nearly under the bed; the latter circumstance indicating that this last attack had been accompanied by severe convulsions.

A third case upon which I observed this phenomenon, is a gentleman of about fifty years, or more, who informed me that he was subject to repeated attacks of chronic pains in the head, frequently accompanied by vertigo, and especially after having had some vexation. In stating these observations I do not mean to imply that the enlarged and tortuous condition of the branches of the temporal artery, as above described, is in all cases accompanied by disease of the smaller blood-vessels of the brain; I merely wish to direct attention to this phenomenon.

The simultaneous appearance of aphasia, accompanying the second attack of our case, directs our attention to that large apoplectic cyst, extending throughout the island of Reil, and, also, to the smaller one in the superior frontal convolution, both of which were lined by a pseudo-membrane, and situated in the left hemisphere. It is obvious that this second attack depended, also, on a capillary hemorrhage into these parts; and which, as the clinical history shows, appears to have taken place by degrees. The accumulation of the blood, slowly escaping from the minute ruptured vessels, gave rise to the formation of that extensive cavity, until it was finally arrested by the pressure of resistance of the surrounding healthy nervous tissues. When first formed it must have been of a greater extent in width than when found after death, a time when the process of reparation had been active for a full

year. The most remarkable feature of this extensive lesion is, that there was never a complete loss of consciousness, the prominent symptoms being only the hemiplegia and the aphasia, accompanied by drowsiness and deranged respiratory function. As the seat of the lesion was in one of those localities of the cerebrum to which, by observation, the faculty of language has been assigned, there was nothing remarkable in the phenomenon of aphasia. More difficult, however, is it to find the true explanation of the paralytic phenomena, manifesting themselves, during this attack, on the right side in the limbs, and on the left side in the face; for, there was no lesion discovered in the corpus striatum, or in those convolutions of the cortex forming the so-called "motor zone" of that hemisphere; neither was any found in the pons varolii, or in the medulla oblongata.

The recent views of a number of prominent pathologists, concerning the pathology of cerebral lesions accompanied with hemiplegia, have been deviating from each other to such an extent, as to render it difficult to the impartial observer to arrive at his own conclusions; more especially, as in support of the different explanations of the phenomena accompanying such lesions, numerous facts have been observed and cited on both sides of the question. As far as regards the case under discussion, it will be seen that, while some of the phenomena are of such a nature as to support the old theory of crossing paralysis, there are others, including those just mentioned, which speak in favor of Dr. Brown-Séquard's recent teachings.

The structure of the pseudo-membrane, revealed by the microscopic examination, is also of interest. From this examination it appeared that it was originally formed and still consisted of the remains of the anatomical elements of the blood, which had been filling the cavity; and that it was not held to the adjacent tissues by any inflammatory product. On the contrary these tissues appeared rather in a state of disintegration, holding, moreover, a considerable number of minute diseased blood-vessels. There remains no doubt but that the second attack was owing to the hemorrhage which gave rise to the cavity. And, as this occurred a whole year

before the death of the patient—during which time the membrane was formed at the surface of the clot by the organization of the fibrin, holding a portion of the colored blood corpuscles, while the rest of the clot was changed into that serous, rusty-colored liquid escaping from the cavity when the section was made—it would be interesting to know how long the patient might still have lived, without the appearance of symptoms caused by the presence of this cavity, and of such a character as to affect his physical condition—if no subsequent hemorrhages had occurred, cutting short his life. And, also, whether, in case of prolonged life, some inflammatory action sufficiently intense to promote the organization of the membrane into a healthy cicatrix, could have been expected to set in. As long as the degenerative process of the minute blood-vessels was not arrested, it is very improbable that such would have been the issue. At any rate, this case, as it was, shows how little the presence of a large apoplectic cavity in the cerebrum may influence the general health of the patient, for the one in question was an eater with a ravenous appetite up to his death.

Little can be said or suggested of the immediate cause of the third and fourth attacks. Although they were marked by loss of consciousness, this was only of a very short duration, and not accompanied by the slightest paralysis, nor by any other symptoms of a serious nature. As the patient recovered so rapidly, both attacks may have been caused by a momentary impediment in the circulation. The loss of consciousness would indicate some momentary disturbance, affecting the cortical layer of the cerebrum. Or, could it be possible that even these short attacks were caused by a minute hemorrhage into the cortical layer, and at the same place in the middle and inferior temporal convolutions where the last and fatal hemorrhage took its seat—and which might thus be regarded as the mere preludes of the fatal issue?

Finally, the extensive hemorrhages in the middle and inferior temporal convolutions of the left hemisphere, and in the thalamus opticus, tegmentum, basis, &c., of the right, must have occurred nearly at the same time. But, as the patient still lived, though unconsciously, more than sixty hours after

the commencement of the fatal attack, it may be presumed that the quantity of effused blood escaped from the vessels during this time very gradually; and as the paralysis of the limbs of the left side was not observed until on the morning following the attack, it is likely that the hemorrhagic effusion into the temporal lobe of the left hemisphere was the first that took place, and which deprived the patient of consciousness; it was soon followed by the other into the right hemisphere, producing the paralysis.

One of the most prominent symptoms in the clinical history of this case, was the aphasia, which appeared with the second attack. The ultimate relation existing between this affection and the mind, has made it an interesting subject of inquiry in human pathology; and it has furnished abundant material to incite the medical mind to a closer investigation of the true mechanism of speech, and the exact mode in which this operates. Not only have the different forms of the disease, with their characteristic symptoms, been thoroughly studied and discussed by some of the most able medical men; but, moreover, the process of language itself has also received its full share of attention. As regards the nature of this process, no great difference seems to exist in the views of different observers; for the main question remaining to be settled, concerns only the localization of this faculty in certain convolutions of the cerebrum. While, for example, some physiologists adopted the view of Broca, who regarded the inferior frontal convolution as the seat of the faculty of speech, others followed Meynert and Sander, and assigned this faculty to the island of Reil.

In the preceding case, the large cavity extending throughout the island of Reil, may, without hesitation, be regarded as the direct cause of the phenomenon of aphasia. And this view seems to be corroborated by the results of the examination of the cyst after death, showing that the improvement in the patient's speech corresponded to the gradual formation of the pseudo-membrane lining the cyst, and protecting the adjacent nervous tissues from farther injury. Thus far this case would speak in favor of the theory of Meynert and Sander, who place the faculty of speech in the island of Reil. The

localization of this faculty in the inferior frontal convolution, or in the island of Reil, seems to be generally admitted by the profession, and is also supported by numerous observations. Nevertheless, it is not an established fact; for the arguments of Dr. Brown-Séquard, in proof of its incorrectness, are certainly strong. As far as the localization of speech is concerned, these arguments appear to be well founded, and worthy to be examined by the unbiased observer; though his theory of functional homogeneousness of the entire cortex cerebri does not appear quite as satisfactory.

In the present case, the patient understood what was said, but could not find the words to express his ideas; and the few words which he articulated were wrong ones. He was not able, as Griesinger once remarked, to find the true image of sound to the image of vision. The nervous current, here, was interrupted while passing through those nerve fibres which connect the cortical centres of vision with those of hearing or sound; when passing, however, from the last named centres to the former, there was no interruption, for the patient was able to transform the words, impressed upon the centre of sound, into images of vision. Moreover, the patient was able to repeat the words which were spoken to him, a phenomenon which may be explained by presuming that the impression made by the spoken words upon the centre of sound, passed, not only to the centre of vision—upon which it left its image—but moreover, without interruption back again to the centre of sound, and thence to those subordinate centres presiding over the muscles of speech, in order to be, finally, transformed into the real, originally spoken words. When the disease was at its height the patient could neither read nor write. Written language, in reality, represents a combination of symbols appreciated only by the organ of vision in the cortex cerebri, upon which they become fixed in the form of visual images, which, by the will of the mind, may be subsequently revived, as long as the brain is in a normal condition. The loss of the understanding of written language, therefore, can be only explained by supposing the existence of disease in those commissural nerve fibres through which the image of a certain object, already fixed upon the mind, becomes associated with

its corresponding symbol, the written word; and of which, also, an impression already exists in that portion of the cortical layer of the cerebrum in which that faculty known as the memory of vision may have its seat.

The above sketch in explanation of the phenomena of aphasia I only drew for the purpose of showing the great complexity of the pathological process concerned in this affection, and the difficulty which attends the exact localization of particular functions in the cortical layer of the brain. In regarding the mind as a force resulting from a combination of the various impressions of external objects upon the substance of the cortical layer of the brain, through the medium of the organs of special sense, it becomes obvious that, in order to localize the faculties of this mental force, the true seat of the terminal elements of the special organs in the cortex cerebri should be known. This, however, is not the case, for, to the extent of my knowledge, no nerve fibre, or bundle of fibres, whether motor or sensory, has ever been traced beyond the corona radiata. Here they are joined by the fibres of the corpus callosum, in the company of which they pursue the rest of their course to the cortical layer. The real difficulty of tracing each set of these fibres separately to their final destination in the cortical layer, is owing to their radiating course, and to the impossibility of distinguishing them from each other. Not knowing, therefore, in what particular regions of the cortex cerebri we may find the terminal elements of the different organs of special sense, it remains impossible to assign a place to particular functions of the brain. Nevertheless, the facts elicited by the experiments of Hitzig, Fritsch, Ferrier and others, together with the pathological observations of Charcot and Pitres in relation to this subject—though, as yet, not explained in a manner to give general satisfaction—may be considered a step farther in this direction. These observations seem to demonstrate the existence of certain motor centres in the cortex cerebri, occupying an intermediate position between the mind and the motor ganglia of the cerebro-spinal axis, and performing the function of transforming the nervous stimuli received from the particular centres of the will, into motor impulses or energy, to be transmitted

through the motor ganglia to the muscles. Thus, the movements of the various groups of muscles of the body would be superintended and regulated through these psycho-motor centres. Although this theory is still far from being firmly established, it is supported by numerous observed facts, pointing, at least, to the probability of its correctness.

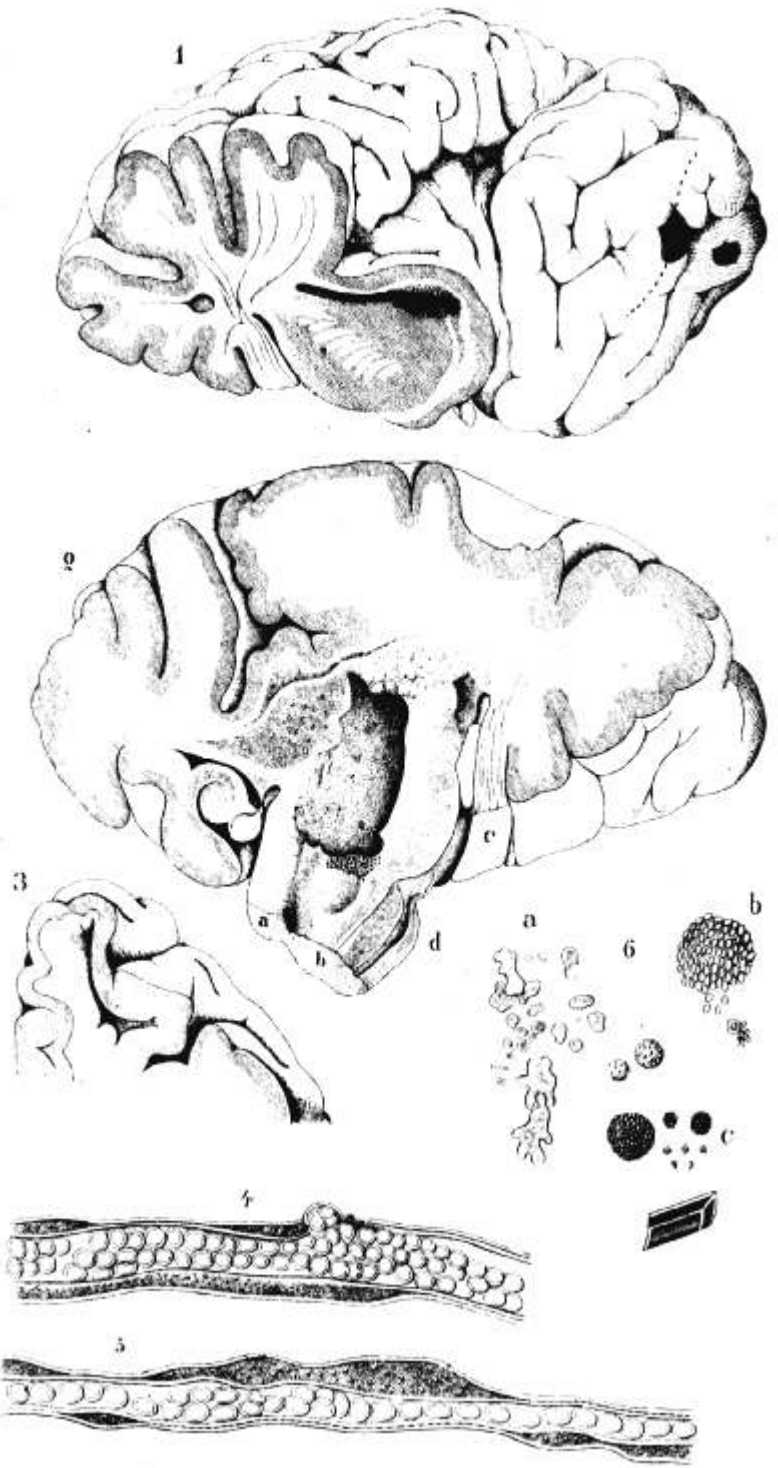
These remarks regarding the localization of the individual mental faculties, are equally applicable to the pretended localization of the faculty of speech. As I have remarked before, language, whether written or spoken, stands in the closest relationship with the special senses, but most especially with those of sight and hearing. In man the great majority of impressions of external objects upon the organ of the mind are made through the instrumentality of these senses; and, according to the comparative intensity of the peripheral stimulus, remain fixed for a shorter or longer period in the form of memory. From this it becomes obvious, that the terminal elements of the organs of vision and sound must extend over a larger region of the cortex cerebri than the third frontal convolution and the island of Reil combined. But as in the majority of cases of aphasia, examined after death, the lesions have been found in, or very near these convolutions, the idea must arise that these parts of the cortex stand, very probably, in some relation with the faculty of speech. And, being situated, at the same time, in close proximity with the so-called "psycho-motor zone," we might venture to regard these convolutions, also, as the psycho-motor centres of the faculty of speech. This supposition may be somewhat corroborated by the view of Meynert, regarding the connection of the claustrum with the nucleus of the auditory nerve. As regards the theory of the localization of the whole apparatus of language in the cortical substance of the third frontal convolution, or of the island of Reil, the arguments of Brown-Séquard, illustrated by his numerous cases, make it untenable. The absence of the island of Reil in the animal brain has been cited in support of this theory. It is not to language in general, however, that this lobe bears any relation, but rather to *articulated* language, a faculty possessed only by man; though it would not be going too far, in regarding even the chattering of the

monkey as a feeble attempt of articulating sounds. All animals, with the possible exception of the lowest classes and orders, possess a symbolic language, by the aid of which they communicate among themselves. In the lower animals these symbols of communication are very simple, consisting only of certain sounds, produced by the rubbing of the legs or wings upon each other, or by some special organs—and, also, in touching each other by means of their antennæ. And, in proof of their perfect understanding of these mutual signs, either by sound or touch, the wonderful actions and labors of the ants and bees bear witness. And, any one who will take both the trouble and pleasure of practically studying the psychology of our domestic animals only, who are constantly near us, as I have been doing for many years, will soon be convinced that they possess a language, consisting in a smaller or greater number of sounds—though not articulated, but varying from each other—by which they express their intentions and desires in the form of distinct ideas. This language, of course, is accompanied by certain expressions of the face, as well as movements made by the tail or other parts of the body.

Explanation of the Illustrations.—Figure 1. Representation of that portion of the left hemisphere of the cerebrum situated directly behind the tuber olfactorium, at which place a transverse vertical section was made. The specimen rests upon the median surface of the hemisphere; the operculum is lifted up, in order to bring the island of Reil into view. The large apoplectic cyst, occupying a considerable portion of the claustrum and capsule, and extending throughout the island of Reil, will be seen upon the cut surface. A smaller cyst is seen near the superior frontal sulcus. Two destructive lesions will be noticed upon the temporal lobe. The larger one represents a cavity, the direction of which is indicated by the dotted line.

Fig. 2. Representation of the posterior surface of a transverse vertical section, made through the right hemisphere, and passing about four millimetres in front of the opening of the aqueduct of Sylvius into the third ventricle. The median surface of the portion of this right hemisphere which the figure represents, will be found slightly bent, a disfiguration caused by the action of the solution of bichromate of potassa, in which the specimen had been lying about a week before this section was made. The large cavity, represented in the figure, also may have had its share in the withdrawal of the crus cerebri from the gyrus hippocampi; *a*, basis; *b*, tegmentum; *c*, median section of corpus callosum; *d*, median section of corpora quadrigemina.

Fig. 3. Representation of the posterior surface of a transverse vertical



From nature by the author

H Klung, Lith. 40 Camp St. New Orleans.

section through the left hemisphere, passing one-half centimetre anterior to the island of Reil, and revealing an apoplectic cyst of a rectangular form, as seen in the figure.

Figs. 4 and 5. Representation of two minute degenerated blood-vessels. In the one (Fig. 4), the formation of a minute aneurism will be observed.

Fig. 6. Component elements of the pseudo-membrane lining the cyst in the island of Reil—*a* and *b*—probable remains of colored blood corpuscles of the clot; *c*, the same elements covered with minute hæmatin crystals, and in company of a large hæmatin crystal.

Figs. 1, 2, and 3, are represented reduced to three-fourths of the natural size. Figs. 4, 5, and 6, are magnified 420 diameters.

ART. II.—TUMOR IN THE CEREBELLUM.

BY S. G. WEBBER, M. D., BOSTON,
LECTURER ON DISEASES OF NERVOUS SYSTEM, HARVARD.

(Read before Boston Society of Medical Observation.)

MR. J. F. M——, æt. 30 years, was seen by request of Dr. W. H. H. Hastings, on August 23. On his father's side consumption was very prevalent. He has never had rheumatism nor venereal disease. Once he had a yellow skin. When five years old he fell over the bannisters and was partially insensible, but soon recovered and never noticed any bad effects from the fall. Last December he first became subject to attacks of dizziness and pain in the head. Before that he had worked hard and late. At times the attacks were severe. Last May he had a sickness which was called a slight attack of diphtheria; after it there was no paralysis, but the dizziness was worse, and the headache was more constant and more severe; it was increased by change of position, especially by lying down after having sat up. There was considerable pain in the eyes; no double vision. Pupils and eyes acted naturally. There was no facial paralysis, no mental disturbance; notwithstanding the very severe pain, the mind was clear almost to the very last. Tongue was protruded straight; there was no tremor of the facial muscles, no exaggerated reflex action.